Head motion: the dirty little secret of neuroimaging in psychiatry

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Psychiatry is at a crossroads when choosing final samples for analysis of neuroimaging data. Many patient populations exhibit significantly increased motion in the scanner compared with healthy controls, suggesting that more patients would need to be excluded to obtain a clean sample. However, this need is often overshadowed by the extensive amount of time and effort required to recruit these valuable and uncommon samples. This commentary sheds light on the impact of motion on imaging studies, drawing examples from psychiatric patient samples to better understand how head motion can confound interpretation of clinically oriented questions. We discuss the impact of even subtle motion artifacts on the interpretation of results as well as how different levels of stringency in quality control can affect findings within nearly identical samples. We also summarize recent initiatives toward harmonization of quality-control procedures as well as tools to prospectively and retrospectively correct for motion artifacts.

Introduction

Neuroimaging studies of psychiatric disorders often face the dilemma of how to handle patient head motion — a dilemma we are reluctant to confront. On one hand, assembling a sufficiently large cohort for meaningful study is a painstaking process, and there is a natural desire to use all the data collected. On the other hand, many patient populations exhibit significantly increased motion in the scanner compared with healthy controls,[1,2] suggesting that more scans must be excluded to obtain a clean enough sample for a significant result. Do common artifacts such as motion really make a difference in large samples? If so, what should be done about it?

Defining motion is deceivingly simple at the core, but it has been surprisingly difficult for researchers to reach consensus over the threshold of acceptable motion that can be tolerated within an MRI study. Moreover, it has become apparent that motion artifacts in neuroimaging research cannot be ignored.[3] Both structural and functional imaging domains are affected by motion artifacts; studies have highlighted that results obtained with the original sample compared with a clean, quality-controlled subset of the data yield significantly different effect sizes and even different neuroanatomical substrates for interpretation. Recently, an editorial by Weinberger and Radulescu[4] brought this issue to the attention of researchers in psychiatry, challenging the common interpretation of findings derived from case–control MRI studies as altered "neurobiology" in patients compared with controls. Instead, the authors urged the field to more critically and carefully acknowledge MRI-derived confounds, such as head motion, that may be clouding key findings in the literature.

In 2002, Blumenthal and colleagues[5] were among the first to report a significant negative association between grey matter brain volumes and severity of motion artifact. This finding is of particular importance in the study of pediatric populations and neurodevelopmental disorders, given children's tendency to be more restless and, in turn, exhibit more movement in the scanner. This realization has rebounded in the literature as researchers attempt to disentangle and reconcile the disparate structural trajectories associated with normal brain development — a critical concept that requires consolidation if we are to broach the topic of abnormal brain trajectories in psychiatric disorders. Nearly a decade ago, Shaw and colleagues[6] conducted a seminal study on the cortical thickness trajectories underlying normal development in a sample ranging from 3.5 to 33 years of age and found predominantly nonlinear associations between cortical thickness and age, with notable peaks within the first decade of life. However, the age window leading up to these peaks of cortical development encompasses an age group (i.e., 5–10 yr) where children have been shown to exhibit the most movement in the MRI scanner.[7] These confounds of movement provide a feasible explanation for the challenge in replicating these findings subsequently in independent samples.[8–12] Ducharme and colleagues[13] addressed such inconsistencies

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using a sample with a similar age range as that in the study by Shaw and colleagues,[6] but with the additional component of three levels of quality control: none, standard and stringent. With increasing stringency of quality control (i.e., removal of more scans with motion), the reported nonlinear cortical thickness associations with age disappeared; instead, predominantly linear associations across most of the brain remained. Characterization of neurodevelopmental trajectories of white matter have also been of great interest and are similarly likely to be significantly influenced by motion. A recent investigation by Roalf and colleagues[14] confirmed this sentiment, showing that significant correlations between commonly reported diffusion metrics (i.e., fractional anisotropy and mean diffusivity) and age are weakened with increased motion estimates.

These examples are convincing evidence that motion artifacts have a significant impact on structural imaging results, but why? As a simple point for comparison, motion in an MRI is akin to motion in any image taken with a camera: the higher degree of motion present, the more blurred and fuzzy the image will be. The basic physics underlying MRI data acquisition add another layer of complexity to the effects of motion or head displacement in a resulting image. When a patient moves in the scanner, it is the spatial frequencies of the MRI, or k-space, that are perturbed. The errors introduced give rise to motion artifacts that are not localized, but propagate throughout the image[3,15] (e.g., ghosting, ringing, blurring). The signal derived from functional MRI (fMRI) can also be greatly perturbed by motion; two detailed reports by Power and colleagues[16,17] describe the complex and variable manner by which different types of motion can impact fMRI acquisitions and increase the proportion of spurious correlations across the brain. In both structural and functional imaging, these artifacts have a downstream impact on all image-derived metrics, such as cortical thickness, regional volumes, or connectivity estimates. Further, what may be more disconcerting are the effects that even subtle motion may have on image quality and subsequent interpretation of commonly used outcome measures, such as cortical thickness.[18] This is particularly true for scans acquired with ultra-high field MRI (i.e., 7 T and above), where longer acquisition times and increased magnetic field strengths confer increased sensitivity to motion.[3] The work of Alexander-Bloch and colleagues[18] emphasized the idea that although our pipelines may have progressed to be able to handle just about anything, they do not necessarily bypass the subtle effects of "micromotion" or biased movements that ultimately may differentiate clinical populations from healthy individuals.

Different levels of stringency in quality control may also yield drastically different results pertaining to clinical questions. A telling example comes from the autism literature, using the openly available Autism Brain Imaging Data Exchange (ABIDE; http://fcon_1000.projects.nitrc.org/indi/abide) dataset, which is a widely used resource to probe questions regarding anatomical trajectories underlying autism-spectrum disorder (ASD), totalling approximately 1100 scans. One investigation by Haar and colleagues[19] found no difference between participants with ASD and typically developing controls, arguing that previous reports of significant findings were likely spurious and not clinically meaningful. However, a recently published study by Khundrakpam and colleagues[20] using this dataset painted a different picture, showing dynamic patterns of significantly thicker cortex in participants with ASD compared with controls across development. So what was the key ingredient separating these two studies? Khundrakpam and colleagues[20] used a stringent quality-control process, excluding half of the available sample; specifically, they excluded about 34% of the sample because of poor-quality scans, which stands in stark contrast to the 4% of poor-quality scans excluded by Haar and colleagues.[19] Given that cortical thinning is a well-replicated consequence of motion[18] and that patients with ASD tend to exhibit more motion in the scanner,[1] it is feasible that the greater degree of motion-affected scans retained in the first sample would have underestimated cortical thickness, particularly within the ASD group, and masked the biologically meaningful signal uncovered by the latter study. The finding of increased thickness in ASD resonates closely with lessons from histology. For instance, postmortem studies have found increased brain volume, alongside disproportionate elevations in both grey and white matter, in individuals with ASD compared with controls,[21,22] supporting the MRI-based findings of Khundrakpam and colleagues.[20]

Cortical thinning and grey matter volume loss have been reported in many psychiatric disorders (for a relevant meta-analysis, see Goodkind and colleagues[23]), although it is difficult to disentangle the degree to which grey matter reductions are due to the underlying neurobiology of the disorder itself or to motion. Yao and colleagues[2] sought this answer in bipolar disorder and schizophrenia samples, finding that reduced surface area and cortical thickness do in fact characterize these psychotic disorders, albeit effect sizes were attenuated when taking head motion into account.

Attention-deficit/hyperactivity disorder (ADHD) poses a particularly interesting challenge in psychiatry, as some of the prominent clinical features characterizing this neurodevelopmental disorder include motion-related symptoms. Two recent studies assessing functional connectivity in ADHD have used rigorous quality-control procedures to more confidently draw upon biologically relevant networks underlying the disorder.[24,25] Mirroring findings in structural imaging, one of these studies also reported reduced effect sizes compared with those reported in previous literature on functional connectivity patterns in ADHD, which the authors largely attribute to their cleaner dataset.[25] Of note, the study by Fair and colleagues[24] demonstrated that correcting for motion can have clinical utility in predicting subtypes of ADHD (i.e., combined v. inattentive subtypes). Specifically, the authors used three different models of motion correction in a machine learning algorithm to classify subgroups of patients with ADHD, finding a relatively high degree of accuracy (i.e., 71%–77%) with two of these models.

One of the largest and most comprehensive investigations examining motion bias across clinical cohorts (i.e., ADHD, ASD and schizophrenia) and different post-processing software was recently conducted by Pardoe and colleagues.[1] This

study used resting-state fMRI acquisitions to inform the degree of movement present in T_1-weighted images and examined this quantitative metric of motion in the context of brain morphometry, namely cortical thickness, grey/white matter contrast and grey matter/subcortical volumes. As expected, motion estimates were higher in all clinical populations than in controls and for the extreme ends of the age distribution (i.e., < 20 yr and > 40 yr). Intriguingly, cortical thickness and measures of cortical contrast were more affected by motion than volumetry, although the latter was affected to a greater degree by the choice of segmentation method. This paints a rather intricate and complex picture of the various levels by which motion may impact neuroimaging analyses of clinical cohorts and underscores the importance of, at minimum, having a clear and consistent quality-assurance protocol to exclude scans visibly affected by motion artifacts. Furthermore, given the manner by which different pipelines may handle motion, it is equally important to study thoroughly the processed outputs — successful runs may not always be linked to trustworthy results.

Many of the examples discussed thus far have come from cross-sectional study designs. However, longitudinal neuroimaging studies are integral in capturing dynamic brain changes that underlie the clinical course of psychiatric illness. Notably, it has been shown previously that head motion shows some degree of test–retest reliability.[26–30] Thus, for longitudinal studies, motion correction should be considered for all time points. It may also be the case that only a subset of longitudinal scans from a single participant are retained for analysis after completing quality control. Fortunately, statistical methods, such as multilevel modelling (i.e., mixed-effects models, hierarchical linear models) are designed to handle missing data points. For a more complete description of these methods, see Singer and Willet.[31]

Guidelines to minimize and correct for head motion artifacts

In the sections that follow, we propose a quality-control workflow that can serve as a framework for both prospective and retrospective datasets. This workflow is depicted in Figure 1. In addition to psychiatric patient samples, it should be noted that the confounds of head motion in the scanner also exist in other fields of biomedical imaging, such as neurology. As such, we propose that this quality-control workflow can be readily generalized to other patient populations, such as patients with neurologic disorders characterized by motor symptoms.

Considerations for prospective datasets

Behavioural training

One of the simplest and arguably most effective techniques in minimizing head motion is to ensure the participant remains as still as possible within the MRI scanner. Sedation and general anesthesia have been successful in minimizing motion, particularly within pediatric populations, but such methods introduce additional confounds and are not always feasible or desirable in most research studies.[32–34] Further, several studies have shown that it is possible to obtain successful scans within pediatric and psychiatric patient samples without the use of more invasive procedures.[7,32,33,35–37] For instance, it is good practice to acclimatize the participant to the MRI setting using a mock scanner and to provide adequate training tailored to the population of interest. If a mock scanner is not available, videos simulating the MRI environment may be used (e.g., http://vimeo.com/32255381).[38] Acclimatizing children to the scanner environment requires more personnel and preparation time than required for adolescents or adults. Detailed pediatric protocols, such as the inclusion of visual aids or allowing the child to view a movie during the scan, have been reported with high success rates.[7,39,40] Neurofeedback paradigms have also been explored, where real-time feedback is provided to participants on their patterns of movement in the scanner,[41] although this technique has had limited returns thus far. In summary, incorporating pre-scan protocols with attention to participant compliance and behaviour during scan acquisition will enhance the likelihood of a successful scan. These points are highlighted in Figure 1, under the "behavioural training" portion of the quality-control workflow.

Technical and methodological considerations

A clear study protocol is required before any scans are collected. The mantra of "quality over quantity" is an important consideration when deciding upon a scanning protocol for a psychiatric neuroimaging study. Focusing on one or two modalities and optimizing their quality (e.g., by acquiring multiple structural imaging scans or functional imaging runs) may be a more efficient use of scan time than acquiring images using many modalities at suboptimal quality. Minimizing motion during scan acquisition may also require technical intervention. In recent years, various research groups have worked diligently toward developing and improving imaging protocols to correct for motion as scans are being acquired in real time. These new techniques hold promise in significantly reducing the proportion of scans that might otherwise need to be excluded owing to motion confounds. For instance, prospective motion correction (PROMO) has been proposed to counter the effects of inevitable participant movement in a proactive manner.[15,42,43] The PROMO framework acts in real time during the acquisition of a scan, where motion can be detected through maintenance of a fixed coordinate system in relation to the participant's position within the scanner, and images are automatically rescanned if significant motion artifact is sensed. This procedure has been shown to drastically reduce the caveats associated with motion in children, and intuitively is expected to hold similar benefits for clinical populations. Framewise integrated real-time MRI monitoring (FIRMM) has also been developed recently to quantify degree of motion from frame to frame as the data are being acquired, allowing MRI technicians and researchers on site to actively monitor head movement accurately and proactively.[44] Similarly, measures of head position and orientation while scanning have been used to improve the quality of images acquired using positron emission tomography (PET).[45] These points can be found in the "technical/methodological" portion of the quality-control workflow in Figure 1.

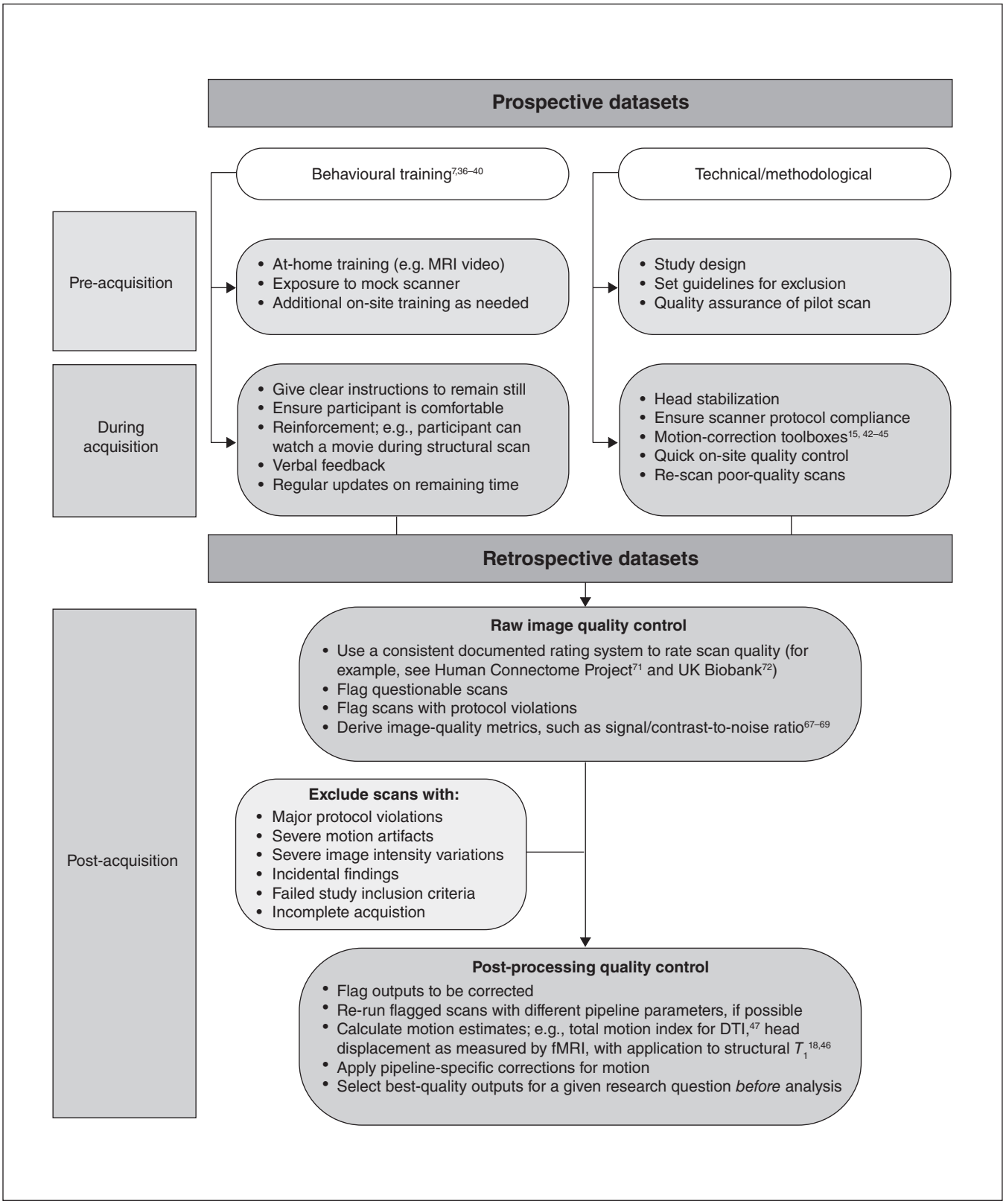

Fig. 1: Proposed quality control workflow for MRI data sets, both prospective and retrospective. Note: this figure is designed as a simplified guide and is not a comprehensive workflow for all imaging modalities and clinical populations. DTI = diffusion tensor imaging; fMRI = functional MRI; QC = quality control.

Considerations for retrospective datasets

The previous section introduced practical suggestions for prospective data collection. A natural question that follows is what solutions exist to handle motion artifacts within the abundance of clinical neuroimaging data that have already been amassed? Having a quantitative measure of motion — a variable that could be used as a means to better match patient and control samples — could be beneficial. We provide some suggestions in Figure 1 that can be applied to raw imaging data to help mitigate the effects of motion artifacts on retrospective datasets.

Correcting for motion after image acquisition

Motion estimates from functional imaging can be used to inform analysis of T_1-weighted structural scans,[18,46] although caution should be exercised when using a proxy measure of motion from another imaging modality. Motion during one acquisition does not necessitate motion during another, even if both acquisitions are collected within the same session. In the realms of diffusion imaging, a total motion index (TMI) has been defined based on several proxies for motion, including calculations of translation, rotation and signal dropout, to obtain a collective "score" of motion.[47] The TMI can then be used as a regressor in the comparison of group differences. Scores of modality-specific toolboxes and workflows have also been developed to help manage the effects of motion on image quality, for application to structural T_1-weighted images,[46,48] diffusion-weighted imaging,[49–53] resting-state[54–58] and task-based fMRI,[59] positron emission tomography[45,60] and arterial spin labelling.[61,62]

Choosing MRI scans for analysis

We have argued the idea that a "clean" dataset, with minimal motion impact, will yield a more biologically valid finding. However, a clear consensus on data cleaning standards has not yet emerged, despite worthy efforts in that direction.[63–65] This is largely attributable to the fact that defining inclusion/exclusion criteria of an MRI scan rests largely upon the research question at hand and on the imaging modality. Standards in fMRI may be more explicit on this point; for instance, Power and colleagues[17] proposed a widely cited method of "scrubbing" fMRI data to remove frames with a high degree of motion and significant amplitude changes in the blood oxygen level–dependent (BOLD) signal. Alternatives to handle different types of motion-related variance in fMRI acquisitions have been described recently by Caballero-Gaudes and Reynolds.[66] Image-quality metrics can also be derived automatically (e.g., signal-to-noise and contrast-to-noise ratios), which provides a quantitative metric that can allow researchers to quickly pinpoint "outliers" that should be flagged (see the "raw image quality control" section of Fig. 1).[67–69] This can be particularly useful when carrying out quality control of large imaging datasets. The critical point is that all quality-control procedures, either on raw images or derived measurements, must be completed prior to any statistical analysis across study participants. There is now an increased sensitivity to the perils of "data fishing," or post hoc analysis (p-hacking), where final published samples are chosen based on the desired result. The field is responding to these issues and, for some journals, preregistration of studies and proposed methods are mandatory.[63,70] It is likely that these initiatives will resonate quickly in the publication sphere. As alluded to earlier, recent initiatives to standardize and consolidate best practices for data analysis and sharing include recommendations for how to handle motion in neuroimaging (http://www.humanbrainmapping.org/COBIDASreport)[65] and active participation in hackathons[64] to reach a consensus on these pressing issues. Finally, many researchers with open datasets are beginning to publish quality-control procedures pertinent to their studies and are encouraging users to follow similar standards; examples include the Human Connectome Project[71] and UK Biobank[72] (see the "raw image quality control" section of Fig. 1). The availability of these large, open datasets will also enable researchers to replicate results and approach the "ground truth" for many questions that still plague the field regarding brain development and alterations in psychiatric disorders. A recent example can be found in the work of Mills and colleauges,[73] where four independent datasets from three different countries were used to examine trajectories of brain development from childhood to early adulthood, finding high replicability across samples. Such replication studies are highly encouraged in the field of psychiatry, especially as more samples of psychiatric patients are being placed in the public domain (e.g., ABIDE,[74,75] ADHD-200 [http://fcon_1000.projects.nitrc.org/indi/adhd200/],[76] the Bipolar-Schizophrenia Network on Intermediate Phenotypes,[77,78] and SchizConnect [http://schizconnect.org/][79]).

Conclusion

The quantification of motion to be accounted for when analyzing data is certainly attractive, compared with mere exclusion of scans, in the analysis of retrospective data. However, sometimes a bad scan is just a bad scan, and it may be worthwhile to exercise the art of "letting go" in severe cases. Neuroimaging technology is developing quickly, and it is reasonable to expect that better algorithms and solutions for handling the blurred edges in our scans will be coming our way. Until then, do not shy away from data cleaning; the rewards gained in validity are worth the loss of a few scans.

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References

1. Pardoe HR, Kucharsky Hiess R, Kuzniecky R. Motion and morphometry in clinical and nonclinical populations. *Neuroimage* 2016;135:177-85.
2. Yao N, Winkler AM, Barrett J, et al. Inferring pathobiology from structural MRI in schizophrenia and bipolar disorder: modeling head motion and neuroanatomical specificity. *Hum Brain Mapp* 2017;38:3757-70.
3. Havsteen I, Ohlhues A, Madsen KH, et al. Are movement artifacts in magnetic resonance imaging a real problem? A narrative review. *Front Neurol* 2017;8:232.
4. Weinberger DR, Radulescu E. Finding the elusive psychiatric "lesion" with 21st-century neuroanatomy: a note of caution. *Am J Psychiatry* 2016;173:27-33.
5. Blumenthal JD, Zijdenbos A, Molloy E, et al. Motion artifact in magnetic resonance imaging: implications for automated analysis. *Neuroimage* 2002;16:89-92.
6. Shaw P, Kabani NJ, Lerch JP, et al. Neurodevelopmental trajectories of the human cerebral cortex. *J Neurosci* 2008;28:3586-94.
7. Greene DJ, Koller JM, Hampton JM, et al. Behavioral interventions for reducing head motion during MRI scans in children. *Neuroimage* 2018;171:234-5.
8. Mills KL, Tamnes CK. Methods and considerations for longitudinal structural brain imaging analysis across development. *Dev Cogn Neurosci* 2014;9:172-90.
9. Aubert-Broche B, Fonov VS, García-Lorenzo D, et al. A new method for structural volume analysis of longitudinal brain MRI data and its application in studying the growth trajectories of anatomical brain structures in childhood. *Neuroimage* 2013;82:393-402.
10. Lebel C, Beaulieu C. Longitudinal development of human brain wiring continues from childhood into adulthood. *J Neurosci* 2011;31:10937-47.
11. Tamnes CK, Walhovd KB, Dale AM, et al. Brain development and aging: overlapping and unique patterns of change. *Neuroimage* 2013;68:63-74.
12. Wierenga LM, Langen M, Oranje B, et al. Unique developmental trajectories of cortical thickness and surface area. *Neuroimage* 2014;87:120-6.
13. Ducharme S, Albaugh MD, Nguyen TV, et al. Trajectories of cortical thickness maturation in normal brain development — the importance of quality control procedures. *Neuroimage* 2016;125:267-79.
14. Roalf DR, Quarmley M, Elliott MA, et al. The impact of quality assurance assessment on diffusion tensor imaging outcomes in a large-scale population-based cohort. *Neuroimage* 2016;125:903-19.
15. White N, Roddey C, Shankaranarayanan A, et al. PROMO: real-time prospective motion correction in MRI using image-based tracking. *Magn Reson Med* 2010;63:91-105.
16. Power JD, Mitra A, Laumann TO, et al. Methods to detect, characterize, and remove motion artifact in resting state fMRI. *Neuroimage* 2014;84:320-41.
17. Power JD, Barnes KA, Snyder AZ, et al. Spurious but systematic correlations in functional connectivity MRI networks arise from subject motion. *Neuroimage* 2012;59:2142-54.
18. Alexander-Bloch A, Clasen L, Stockman M, et al. Subtle in-scanner motion biases automated measurement of brain anatomy from in vivo MRI. *Hum Brain Mapp* 2016;37:2385-97.
19. Haar S, Berman S, Behrmann M, et al. Anatomical abnormalities in autism? *Cereb Cortex* 2016;26:1440-52.
20. Khundrakpam BS, Lewis JD, Kostopoulos P, et al. Cortical thickness abnormalities in autism spectrum disorders through late childhood, adolescence, and adulthood: a large-scale MRI study. *Cereb Cortex* 2017;27:7211-31.
21. Casanova MF. The neuropathology of autism. *Brain Pathol* 2007;17:422-33.
22. Courchesne E, Redcay E, Kennedy DP. The autistic brain: birth through adulthood. *Curr Opin Neurol* 2004;17:489-96.
23. Goodkind M, Eickhoff SB, Oathes DJ, et al. Identification of a common neurobiological substrate for mental illness. *JAMA Psychiatry* 2015;72:305.
24. Fair DA, Nigg JT, Iyer S, et al. Distinct neural signatures detected for ADHD subtypes after controlling for micro-movements in resting state functional connectivity MRI data. *Front Syst Neurosci* 2013;6:80.
25. Mostert JC, Shumskaya E, Mennes M, et al. Characterising resting-state functional connectivity in a large sample of adults with ADHD. *Prog Neuropsychopharmacol Biol Psychiatry* 2016;67:82-91.
26. Zuo X-N, Anderson JS, Bellec P, et al. An open science resource for establishing reliability and reproducibility in functional connectomics. *Sci Data* 2014;1:140049.
27. Van Dijk KRA, Sabuncu MR, Buckner RL. The influence of head motion on intrinsic functional connectivity MRI. *Neuroimage* 2012;59:431-8.
28. Yan C-G, Cheung B, Kelly C, et al. A comprehensive assessment of regional variation in the impact of head micromovements on functional connectomics. *Neuroimage* 2013;76:183-201.
29. Kong XZ, Zhen Z, Li X, et al. Individual differences in impulsivity predict head motion during magnetic resonance imaging. *PLoS One* 2014;9:e104989.
30. Liu W, Wei D, Chen Q, et al. Longitudinal test-retest neuroimaging data from healthy young adults in southwest China. *Sci Data* 2017;4:170017.
31. Singer JD, Willet JB. *Applied longitudinal data analysis: modeling change and event occurrence*. New York: Oxford University Press, 2003.
32. Nordahl CW, Simon TJ, Zierhut C, et al. Brief report: methods for acquiring structural MRI data in very young children with autism without the use of sedation. *J Autism Dev Disord* 2008;38:1581-90.
33. Edwards AD, Arthurs OJ. Paediatric MRI under sedation: Is it necessary? What is the evidence for the alternatives? *Pediatr Radiol* 2011;41:1353-64.
34. Reuter M, Tisdall MD, Qureshi A, et al. Head motion during MRI acquisition reduces gray matter volume and thickness estimates. *Neuroimage* 2015;107:107-15.
35. Rosenberg DR, Sweeney JA, Gillen JS, et al. Magnetic resonance imaging of children without sedation: preparation with simulation. *J Am Acad Child Adolesc Psychiatry* 1997;36:853-9.
36. Nordahl CW, Mello M, Shen AM, et al. Methods for acquiring MRI data in children with autism spectrum disorder and intellectual impairment without the use of sedation. *J Neurodev Disord* 2016;8:20.
37. Greene DJ, Black KJ, Schlaggar BL. Considerations for MRI study design and implementation in pediatric and clinical populations. *Dev Cogn Neurosci* 2016;18:101-12.
38. Mills KL, Tamnes CK. Methods and considerations for longitudinal structural brain imaging analysis across development. *Dev Cogn Neurosci* 2014;9:172-90.
39. Barnea-Goraly N, Weinzimer SA, Ruedy KJ, et al. High success rates of sedation-free brain MRI scanning in young children using simple subject preparation protocols with and without a commercial mock scanner — the Diabetes Research in Children Network (DirecNet) experience. *Pediatr Radiol* 2014;44:181-6.

40. Centeno M, Tierney TM, Perani S, et al. Optimising EEG-fMRI for localisation of focal epilepsy in children. *PLoS One* 2016;11: e0149048.
41. Lal TM, Baldwin PR, Cai M-B, et al. Real time functional MRI training to decrease motion in imaging studies: lack of significant improvement. *Bull Menninger Clin* 2016;80:348-56.
42. Brown TT, Kuperman JM, Erhart M, et al. Prospective motion correction of high-resolution magnetic resonance imaging data in children. *Neuroimage* 2010;53:139-45.
43. Tisdall MD, Reuter M, Qureshi A, et al. Prospective motion correction with volumetric navigators (vNavs) reduces the bias and variance in brain morphometry induced by subject motion. *Neuroimage* 2016;127:11-22.
44. Dosenbach NUF, Koller JM, Earl EA, et al. Real-time motion analytics during brain MRI improve data quality and reduce costs. *Neuroimage* 2017;161:80-93.
45. Menke M, Atkins MS, Buckley KR. Compensation methods for head motion detected during PET imaging. *IEEE Trans Nucl Sci* 1996;43:310-7.
46. Savalia NK, Agres PF, Chan MY, et al. Motion-related artifacts in structural brain images revealed with independent estimates of in-scanner head motion. *Hum Brain Mapp* 2017;38:472-92.
47. Yendiki A, Koldewyn K, Kakunoori S, et al. Spurious group differences due to head motion in a diffusion MRI study. *Neuroimage* 2014;88:79-90.
48. Backhausen LL, Herting MM, Buse J, et al. Quality control of structural MRI images applied using FreeSurfer — a hands-on workflow to rate motion artifacts. *Front Neurosci* 2016;10:558.
49. He X, Liu W, Li X, et al. Automated assessment of the quality of diffusion tensor imaging data using color cast of color-encoded fractional anisotropy images. *Magn Reson Imaging* 2014;32:446-56.
50. Oguz I, Farzinfar M, Matsui J, et al. DTIPrep: quality control of diffusion-weighted images. *Front Neuroinform* 2014;8:4.
51. Farzinfar M, Oguz I, Smith RG, et al. Diffusion imaging quality control via entropy of principal direction distribution. *Neuroimage* 2013;82:1-12.
52. Andersson JLR, Graham MS, Zsoldos E, et al. Incorporating outlier detection and replacement into a non-parametric framework for movement and distortion correction of diffusion MR images. *Neuroimage* 2016;141:556-72.
53. Yendiki A, Koldewyn K, Kakunoori S, et al. Spurious group differences due to head motion in a diffusion MRI study. *Neuroimage* 2014;88:79-90.
54. Yan C-G, Wang X-D, Zuo X-N, et al. DPABI: data processing & analysis for (resting-state) brain imaging. *Neuroinformatics* 2016;14:339-51.
55. Patriat R, Reynolds RC, Birn RM. An improved model of motion-related signal changes in fMRI. *Neuroimage* 2017;144:74-82.
56. Patriat R, Molloy EK, Birn RM. Using edge voxel information to improve motion regression for rs-fMRI connectivity studies. *Brain Connect* 2015;5:582-95.
57. Pruim RHR, Mennes M, Buitelaar JK, et al. Evaluation of ICA-AROMA and alternative strategies for motion artifact removal in resting state fMRI. *Neuroimage* 2015;112:278-87.
58. Pruim RHR, Mennes M, van Rooij D, et al. ICA-AROMA: a robust ICA-based strategy for removing motion artifacts from fMRI data. *Neuroimage* 2015;112:267-77.
59. Tierney TM, Weiss-Croft LJ, Centeno M, et al. FIACH: a biophysical model for automatic retrospective noise control in fMRI. *Neuroimage* 2016;124:1009-20.
60. Green MV, Seidel J, Stein SD, et al. Head movement in normal subjects during simulated PET brain imaging with and without head restraint. *J Nucl Med* 1994;35:1538-46.
61. Shirzadi Z, Crane DE, Robertson AD, et al. Automated removal of spurious intermediate cerebral blood flow volumes improves image quality among older patients: a clinical arterial spin labeling investigation. *J Magn Reson Imaging* 2015;42:1377-85.
62. Wang Z. Improving cerebral blood flow quantification for arterial spin labeled perfusion MRI by removing residual motion artifacts and global signal fluctuations. *Magn Reson Imaging* 2012;30:1409-15.
63. Gorgolewski KJ, Poldrack RA, Carp J, et al. A practical guide for improving transparency and reproducibility in neuroimaging research. *PLoS Biol* 2016;14:e1002506.
64. Craddock R, Margulies DS, Bellec P, et al. Brainhack: a collaborative workshop for the open neuroscience community. *Gigascience* 2016;5:16.
65. Nichols TE, Das S, Eickhoff SB, et al. Best practices in data analysis and sharing in neuroimaging using MRI. *Nat Neurosci* 2017;20:299-303.
66. Caballero-Gaudes C, Reynolds RC. Methods for cleaning the BOLD fMRI signal. *Neuroimage* 2017;154:128-49.
67. Magnotta VA, Friedman L. FIRST BIRN. Measurement of signal-to-noise and contrast-to-noise in the fBIRN Multicenter Imaging Study. *J Digit Imaging* 2006;19:140-7.
68. Dietrich O, Raya JG, Reeder SB, et al. Measurement of signal-to-noise ratios in MR images: influence of multichannel coils, parallel imaging, and reconstruction filters. *J Magn Reson Imaging* 2007;26:375-85.
69. Esteban O, Birman D, Schaer M, et al. MRIQC: advancing the automatic prediction of image quality in MRI from unseen sites. *PLoS One* 2017;12:e0184661.
70. Gonzales JE, Cunningham CA. The promise of pre-registration in psychological research. *Am Psychol Assoc Psychol Sci Agenda* 2015; 1. Available: www.apa.org/science/about/psa/2015/08/pre-registration.aspx (accessed 2018 Jan 9).
71. Marcus DS, Harms MP, Snyder AZ, et al. Human Connectome Project informatics: quality control, database services, and data visualization. *Neuroimage* 2013;80:202-19.
72. Alfaro-Almagro F, Jenkinson M, Bangerter NK, et al. Image processing and quality control for the first 10,000 brain imaging datasets from UK Biobank. *Neuroimage* 2018;166:400-24.
73. Mills KL, Goddings A-L, Herting MM, et al. Structural brain development between childhood and adulthood: convergence across four longitudinal samples. *Neuroimage* 2016;141:273-81.
74. Di Martino A, Yan C-G, Li Q, et al. The autism brain imaging data exchange: towards a large-scale evaluation of the intrinsic brain architecture in autism. *Mol Psychiatry* 2014;19:659-67.
75. Di Martino A, O'Connor D, Chen B, et al. Enhancing studies of the connectome in autism using the autism brain imaging data exchange II. *Sci Data* 2017;4:170010.
76. Milham M, Fair D, Mennes M, et al.; The ADHD-200 Consortium. a model to advance the translational potential of neuroimaging in clinical neuroscience. *Front Syst Neurosci* 2012;6:62.
77. Book GA, Stevens MC, Assaf M, et al. Neuroimaging data sharing on the neuroinformatics database platform. *Neuroimage* 2016; 124:1089-92.
78. Tamminga CA, Ivleva EI, Keshavan MS, et al. Clinical phenotypes of psychosis in the Bipolar-Schizophrenia Network on Intermediate Phenotypes (B-SNIP). *Am J Psychiatry* 2013;170:1263-74.
79. Wang L, Alpert KI, Calhoun VD, et al. SchizConnect: mediating neuroimaging databases on schizophrenia and related disorders for large-scale integration. *Neuroimage* 2016;124:1155-67.